Humid heat acclimation does not elicit a preferential sweat redistribution toward the limbs

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Patterson, Mark J., Jodie M. Stocks, and Nigel A. S. Taylor. Humid heat acclimation does not elicit a preferential sweat redistribution toward the limbs. Am J Physiol Regul Integr Comp Physiol 286: R512–R518, 2004. First published October 24, 2003; 10.1152/ajpregu.00359.2003.—We tested the hypothesis that local sweat rates would not display a systematic postadaptation redistribution toward the limbs after humid heat acclimation. Eleven nonadapted males were acclimated over 3 wk (16 exposures), cycling 90 min/day, 6 days/wk (40°C, 60% relative humidity), using the controlled-hyperthermia acclimation technique, in which work rate was modified to achieve and maintain a core temperature (38.5°C). Local sudomotor adaptation (forehead, chest, scapula, forearm, thigh) and onset thresholds were studied during constant work intensity heat stress tests (39.8°C, 59.2% relative humidity) conducted on days 1, 8, and 22 of acclimation. The mean body temperature (Tb) at which sweating commenced (threshold) was reduced on days 8 and 22 (P < 0.05), and these displacements paralleled the resting thermoneutral Tb shift, such that the Tb change to elicit sweating remained constant from days 1 to 22. Whole body sweat rate increased significantly from 0.87 ± 0.06 l/h on day 1 to 1.09 ± 0.08 l/h on days 8 and 22, respectively. However, not all skin regions exhibited equivalent relative sweat rate elevations from day 1 to day 22. The relative increase in forearm sweat rate (117 ± 31%) exceeded that at the forehead (47 ± 18%; P < 0.05) and thigh (42 ± 16%; P < 0.05), while the chest sweat rate elevation (106 ± 29%) also exceeded the thigh (P < 0.05). Two unique postacclimation observations arose from this project. First, reduced sweat thresholds appeared to be primarily related to a lower resting Tb, and more dependent on Tb change. Second, our data did not support the hypothesis of a generalized and preferential trunk-to-limb sweat redistribution after heat acclimation.

The principal avenue for heat dissipation in hot environments is via evaporative cooling, with sudomotor enhancement accompanying endurance training and heat adaptation. The latter can elicit a reduced core temperature (Tc) threshold for sweating onset (6, 8, 24, 37), a greater sensitivity to changes in Tc (13, 32, 40), an elevated steady-state expulsion rate (24–26, 37), eccrine gland hypertrophy (33), and an apparent redistribution of sweating toward the limbs (15, 31, 37), representing our potentially most potent adaptive responses to chronic heat stress. In this paper, we focus on sudomotor threshold and local sweat rate (mω) changes accompanying humid heat acclimation.

Höfler (15) and Shvartz et al. (37) first reported heat acclimation induced a peripheral redistribution of sweating, such that postacclimation limb mω appeared to be elevated more than at central body sites. This apparent peripheral shift in secretion could facilitate greater heat dissipation, if lower preacclimation limb mω (5, 12, 19) was also associated with less than optimal local evaporation rates. Because limbs have a relatively large surface area:mass ratio, an elevation in sweating and evaporation could enhance thermal homeostasis. However, while data from Höfler (15), Shvartz et al. (37), and Regan et al. (31) are consistent with a redistribution of sweat, closer inspection revealed methodological limitations that may invalidate that interpretation.

First, these observations were based on data collected from only two to three skin sites (31, 37) and not from simultaneous measures from the head, torso, and limbs. Second, the observations of Höfler (15) and Shvartz et al. (37) were not statistically analyzed. Thus, between-site differences in sweat secretion were reported entirely in descriptive terms and are difficult to use for comparing sudomotor function across skin regions. Finally, local skin pressure elicits sudomotor measurement artefact (28, 29), and because Höfler (15), Shvartz et al. (37), and Regan et al. (31) used methods that resulted in local pressure application, their data may have suffered pressure hidrosis effects, rendering between-site comparisons difficult. In view of these limitations, we tested the veracity of a postacclimation peripheral sweat redistribution accompanying a 21-day humid heat acclimation regimen, using five simultaneous measures of local mω.

Thermoregulatory thresholds define the body temperature at which a thermoeffector mechanism is recruited. These thresholds display considerable plasticity and are readily influenced by thermal stimuli outside the thermoneutral zone, with a reduced sweating threshold typically accompanying heat acclimation (6, 8, 24, 37). A reduction in resting body temperature is often observed as acclimation progresses. Buono et al. (3) were perhaps the first to suggest that the reduced sweating threshold accompanying heat acclimation may be associated with a lower resting body temperature. This presumption was extrapolated from Fox et al. (8) and Shvartz et al. (37), although neither group described the relationship. Likewise, data from Stephenson et al. (39) indicate parallel reductions of resting body and sweat threshold temperatures over the circadian cycle. Therefore, it is possible that, rather than being primarily dictated by the existence of a simple body temperature threshold, the initiation of sweating may also be driven by body temperature displacement. We provide evidence consistent with this possibility.

In the current experiment, we aimed to evaluate the distribution of local sudomotor changes and sweat thresholds during...
repeated exposure to humid heat. We tested the hypothesis that local $m_{nw}$ would not display a systematic postadaptation redistribution toward the limbs. Herein, we provide evidence that challenges the notion of an acclimation-induced preferential sweat redistribution to the limbs, while exploring possible mechanisms underlying differential changes in local $m_{nw}$. We also report data that are consistent with the hypothesis that the postacclimation sweat threshold may not necessarily result from a fixed combination of core and skin thermoafferent information but may instead be sensitive to the changes in body temperature, particularly that of the body core.

METHODS

Subjects. Eleven healthy, physically active males [age 21.6 yr (range 18–28 yr); mass 78.2 kg (59.3–87.9 kg); peak oxygen consumption 4.2 l/min (3.3–6.7 l/min; semi-recumbent cycling)] without a previous history of heat acclimation were acclimated during August–October (Southern Hemisphere winter–spring). The local minimum and maximum temperatures for the preceding June–September period range from 8.4 to 10.6°C and 16.9 to 20.1°C, respectively. No control group was employed because this investigation examined sudomotor function, as affected by heat acclimation, rather than the effectiveness of the heat acclimation protocol. All procedures were approved by the Human Research Ethics Committee, University of Wollongong, and subjects provided written, informed consent.

Experimental design. Heat acclimation was induced using 16 treatment exposures (exercise and heat stress) and assessed using three standardized heat stress tests (HSTs). During heat acclimation, we used the controlled-hyperthermia technique (7), in which work rate was modified so that subjects achieved and maintained a target $T_c$ (38.5°C) for at least 60 min of the heat exposure. This method differs from the constant workload regimen and results in sustained thermal strain rather than a progressive decline in strain as acclimation progresses. Sudomotor function was assessed during HSTs conducted at the same time of day (within and between subjects) before (day 1) and after 1 wk (day 8) and 3 wk (day 22) of humid heat acclimation.

For each HST, subjects arrived at the laboratory in a fasted and euhydrated state and were served a high-carbohydrate breakfast (38 kJ/kg) with supplementary fluid (10 ml/kg) and then rested for 4 h (seated). At least 90 min before the commencement of each HST, subjects were moved to the climate chamber (28°C, 60% relative humidity), and each HST commenced with a baseline, resting data collection, after which chamber temperature was elevated over ~10 min. Subjects rested in these conditions for a further 30 min [39.8 ± 0.5°C, relative humidity 59.2 ± 0.8% (SD), and wind speed <0.5 m/s], then commenced three 30-min periods of semicorement cycling (90 min), each at ~30% peak work rate ($W_{peak}$), with 2 min rest after 30 and 60 min. The relative exercise intensity was maintained across the three HSTs because Sawka et al. (35) earlier reported an improved work efficiency after heat acclimation. Thus postacclimation changes in $T_c$ elevation could be attributed to either elevated heat dissipation or reduced heat production, unless work rate was modulated. Peak oxygen consumption and work rate were assessed before days 1 and 22, as it was assumed that exercise efficiency would not improve over six treatments (day 8). While peak oxygen consumption was not significantly increased on day 22 (4.2 ± 0.3 vs. 4.3 ± 0.4 l/min; $P > 0.05$), $W_{peak}$ was increased by 10 W (0–30 W) ($P < 0.05$). Consequently, the mean work rate was greater on day 22 (103 ± 4 vs. 106 ± 4 W; $P < 0.05$).

On six occasions during the first HST (day 1), $T_c$ would have exceeded 39.5°C before completion of the 90-min work bout, so subsequent work rates were reduced after 30 and 60 min so that each subject completed 90 min with $T_c \leq 39.5°C$. The average relative work rates for these 30-min periods were 28.9% (±1.8; SD), 27.5% (±3.2), and 26.6% (±3.6) $W_{peak}$ (respectively). At the end of 90 min, subjects rested for 2 min before commencing a ramp forcing function to volitional fatigue (4% $W_{peak}$/min), or $T_c \geq 39.5°C$.

Euhydration was confirmed immediately before the commencement of each HST using measurements of plasma (277 ± 2 mosmol/kgH$_2$O; SD) and urine (406 ± 151 mosmol/kgH$_2$O; SD) osmolality. Plasma osmolality was in the expected euhydration range for the vapor-pressure technique (model 5100C, Wescor) and was consistent with previous data from other laboratories using this technique (10). Fluid replacement was not permitted during HSTs, although at the completion of each HST, subjects replaced 100% of fluid loss (mass change) with an isosmotic drink.

Treatment (acclimation) day used the controlled-hyperthermia technique to achieve a target $T_c$ of 38.5°C. Each day, subjects cycled upright (Monark 868 ergometer) for 90 min in a hot-humid environment (40°C, 60% relative humidity), except for three rest days (days 7, 14, 21). In each session, subjects commenced cycling at a work rate sufficient to elevate $T_c$ to 38.5°C within ~30 min (~44% $W_{peak}$); thereafter work rate was adjusted to maintain the constant and elevated $T_c$ target (after Ref. 31). Subjects rested for 2 min every 30 min, at which time 200 ml of water was consumed. At the end of the 90-min acclimation bout, body mass loss was reduced by an average of 2.49% (range 1.39–3.62), and subjects were rehydrated with an isosmotic drink, consuming the equivalent of 100% of the body mass change before leaving the laboratory. Exposures were reduced to 60 min on day 20, due to the reassessment of peak aerobic power before each exercise-heat exposure.

During treatment (acclimation) exposures, $T_c$ was monitored at the auditory canal and rectum. Both measures exhibit a close correlation with esophageal temperature during moderate exercise in the heat (5). Peak aerobic power tests involved semicorement cycling to volitional fatigue, using a ramp forcing function (3 W/s; Excalibur, Lode BV), with expired gases collected and analyzed to derive peak oxygen consumption (2900 Sensormedics).

Physiological measures during HSTs. Esophageal ($T_e$) and skin temperatures were recorded continuously during each HST. An esophageal thermistor (Edale Instruments) was inserted transnasally (~40 cm; after Ref. 22). Skin temperatures were measured at eight sites using surface thermistors (Yellow Springs Instruments, EU mini thermistor), attached with a single layer of waterproof tape, and mean skin temperature ($T_{SK}$) was calculated using an area-weighted mean (18). All thermistors were calibrated across the physiological temperature range against a certified reference mercury thermometer (Dobros total immersion thermometer, Dobie Instruments). Both esophageal and skin temperatures were recorded at 0.2 Hz using a portable data logger (Grant Instruments, 1206 Series Squared). Mean body temperature ($T_B$) was derived as 0.9$T_{es} + 0.1T_{sk}$ (11, 23). Cardiac frequency was monitored from ventricular depolarization (Polar Electro Sports Tester).

Local $m_{nw}$ were measured simultaneously using ventilated sweat capsules (3.16 ± 0.05 cm$^2$) attached to five skin sites: forehead (middle), chest (~7 cm superior to nipple and ~7 cm lateral from sternum), scapula (immediately superior to the scapula spine and ~15 cm lateral to vertebral column), forearm (dorsal surface, immediately distal to elbow), and thigh (equidistant between knee and hip on ventral surface). To avoid pressure-hidrosis, sweat capsules were constructed with flanged perimeters, to which an adhesive was applied (Collodion, Mavidon Medical Products), creating an air-tight seal with the skin. Air ventilating each capsule was initially passed over a saturated solution of lithium chloride (0.6–1.2 l/min) of a constant, known temperature. The relative humidities (capacitance hygrometry) and temperatures of the postcapsular air were measured (Multi-Site Sweat Monitor, Clinical Engineering Solutions) and sampled at 0.2 Hz (Metrabyte DAS 1602); $m_{nw}$ were derived from changes in the relative humidity and temperature of air entering and leaving each sweat capsule. Hygrometers were calibrated using saturated solution standards of lithium chloride and sodium chloride. Whole body
sweating was derived from body mass changes, uncorrected for respiratory or metabolic variations (fw-150k, A&D).

While local $m_{sw}$ was monitored continuously throughout each HST, the local $m_{sw}$ after 60 min of exercise was used to compare regional differences in sudomotor function between HSTs because $m_{sw}$ had generally plateaued at this time. The sweat thresholds (onset) were defined as the time points after commencement of resting heat stress, beyond which local $m_{sw}$ was elevated, and maintained above baseline, for at least 5 min (i.e., established sweating). The corresponding body temperatures at these times were defined as the threshold temperatures, which occurred within the initial 40 min of resting heat stress (94% of all cases), and before the commencement of exercise. Subtracting the $T_b$ and $T_w$ at the thresholds from the thermoneutral resting $T_b$ and $T_w$ provided the changes in $T_b$ and $T_w$ needed to elicit sweating. Sudomotor sensitivity (gain; $\Delta m_{sw}/\Delta T_{sw}$) was calculated over a 5-min period, starting at 3 min after the commencement of exercise. This was achieved by fitting a linear function to the relationship between $m_{sw}$ and $T_w$ with the gradient of this relationship corresponding with sweat sensitivity.

Statistical analysis. Statistical analyses were performed on data from days 1, 8, and 22 using a two-way ANOVA, with repeated measures. One-way ANOVAs were subsequently performed to isolate differences between days at separate time points and to isolate differences between time points within a day. Sources of significant difference were isolated using Tukey’s HSD statistic; $\alpha$ was set at 5% level for all analyses. Data are presented as means with SEs, unless otherwise stated.

RESULTS

The current protocol elicited a significant heat acclimation across the 3 wk. This was confirmed by the reduced exercise and resting cardiac frequencies (Table 1), $T_{es}$, $T_{sk}$, and $T_b$ (Fig. 1), the elevated total work performed during each HST, and the increased whole body $m_{sw}$ (Table 1). Because we employed an increasing exercise forcing function from days 1 to 22, these changes are of considerable physiological significance.

The mean thermoneutral $T_{es}$ after 4 h of rest, was reduced from 36.91°C on day 1 by 0.20°C on day 8 and by 0.32°C on day 22 ($P < 0.05$; Fig. 1A), while no difference was evident between days 8 and 22 ($P > 0.05$). The resting $T_{sk}$ did not differ between days ($P > 0.05$; Fig. 1B). Therefore, reductions in the thermoneutral resting $T_b$ on days 8 and 22 (0.19°C and 0.30°C, $P < 0.05$; Fig. 1C) primarily reflected $T_{es}$ changes.

The preexposure $T_{es}$ offset accompanying acclimation and observed at rest was essentially maintained throughout exercise on days 8 and 22 (Fig. 1A). That is, $T_{es}$ differences between HSTs, particularly on day 8, largely reflected the offsetting of the resting $T_{es}$. However, after 90 min of exercise, the $T_{es}$ on day 22 was significantly lower than observed on days 1 (0.57°C; $P < 0.05$) and 8 (0.40°C; $P < 0.05$), while differences between days 1 and 8 were insignificant ($P > 0.05$). In general, $T_{sk}$ was lower during the HST on days 8 and 22, although no differences were observed between these days ($P > 0.05$), and the $T_b$ response exhibited similar trends to $T_{es}$ (Fig. 1C). The core-skin temperature gradient during exercise was unaffected by heat acclimation ($P > 0.05$), with the mean gradients being 0.54°C ±0.04 on day 1, 0.71°C ±0.06 on day 8, and 0.54°C ±0.03 on day 22.

Sweat recruitment preceded the initiation of exercise, occurring during heated rest in most HSTs (Fig. 2A) and exhibited the following caudal-to-rostral pattern: thigh (first), then forehead, scapula, chest, and forearm ($P < 0.05$; Fig. 2A). Heat acclimation did not influence this recruitment pattern ($P > 0.05$; Fig. 2A). However, the site-specific $T_{sk}$ thresholds for sweating onset were reduced on both days 8 and 22 ($P < 0.05$; Fig. 2B), but no differences were evident between these days ($P > 0.05$). Because the $T_{sk}$ was not different between HSTs at the initiation of sweating ($P > 0.05$; Table 2), the reduced $T_{es}$, which paralleled its preexposure offset, was primarily responsible for these lower $T_{sk}$ thresholds ($P < 0.05$; Table 2), such that the displacement in $T_{sk}$ required to initiate sweating was equivalent between HSTs ($P > 0.05$; Fig. 2C).

Table 1. Resting and exercise heart rates, whole body sweat rates, and total work performed on days 1, 8, and 22 of humid heat acclimation

<table>
<thead>
<tr>
<th>Day 1</th>
<th>Day 8</th>
<th>Day 22</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac frequency, beats/min ($n = 10$)</td>
<td>74 ±1</td>
<td>69 ±2*</td>
</tr>
<tr>
<td>Exercise 60 min</td>
<td>150 ±4</td>
<td>140 ±4*</td>
</tr>
<tr>
<td>Whole body sweat rate, l/h</td>
<td>0.87 ±0.06</td>
<td>1.09 ±0.08*</td>
</tr>
<tr>
<td>Total work performed, kJ</td>
<td>552 ±36</td>
<td>602 ±31</td>
</tr>
</tbody>
</table>

Values are means ± SE; $n = 11$ unless otherwise stated. *Significantly different from day 1.
change in $T_{es}$ threshold did not differ between HSTs ($P > 0.05$; Table 2). Therefore, because $T_{sk}$ did not differ between HSTs, either during thermoneutral rest or at the initiation of sweating, it seemed that the diminished resting and sweat threshold $T_{th}$ were reliant on the $T_{es}$ reduction.

Short-term heat acclimation (day 8) did not affect the sensitivity (gain) of the sweating response to an increase in $T_b$ ($P > 0.05$; Fig. 3C). However, on day 22, the sweat sensitivity was greater than on day 1 for all skin regions except the scapula ($P < 0.05$; Fig. 3C). While it did appear that sudomotor sensitivity was greater on day 22 compared with day 8, these differences only reached significance for the forehead ($P < 0.05$; Fig. 3C).

### Table 2. $T_{sk}$ and $T_{es}$ at the sweat threshold and the change in $T_{sk}$ and $T_{es}$ needed to initiate sweating on days 1, 8, and 22 of humid heat acclimation

<table>
<thead>
<tr>
<th></th>
<th>Thigh</th>
<th>Forehead</th>
<th>Scapula</th>
<th>Chest</th>
<th>Forearm</th>
</tr>
</thead>
<tbody>
<tr>
<td>$T_{sk}$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Day 1</td>
<td>34.72±0.30</td>
<td>35.12±0.28</td>
<td>36.01±0.23</td>
<td>35.84±0.15</td>
<td>35.95±0.16</td>
</tr>
<tr>
<td>Day 8</td>
<td>34.60±0.36</td>
<td>35.16±0.36</td>
<td>35.29±0.31</td>
<td>35.53±0.21</td>
<td>35.63±0.21</td>
</tr>
<tr>
<td>Day 22</td>
<td>34.91±0.41</td>
<td>35.43±0.26</td>
<td>35.66±0.28</td>
<td>35.60±0.25</td>
<td>35.74±0.25</td>
</tr>
<tr>
<td>$\Delta T_{sk}$ sweat recruitment, °C</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Day 1</td>
<td>2.09±0.28</td>
<td>2.49±0.22</td>
<td>3.37±0.30</td>
<td>3.21±0.22</td>
<td>3.32±0.19</td>
</tr>
<tr>
<td>Day 8</td>
<td>2.07±0.40</td>
<td>2.63±0.41</td>
<td>3.76±0.36</td>
<td>3.01±0.26</td>
<td>3.11±0.27</td>
</tr>
<tr>
<td>Day 22</td>
<td>2.46±0.41</td>
<td>2.98±0.29</td>
<td>3.21±0.32</td>
<td>3.15±0.29</td>
<td>3.29±0.29</td>
</tr>
<tr>
<td>$T_{es}$</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Day 1</td>
<td>36.98±0.06</td>
<td>36.99±0.06</td>
<td>37.02±0.06</td>
<td>37.03±0.06</td>
<td>37.05±0.07</td>
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<tr>
<td>Day 8</td>
<td>36.76±0.06</td>
<td>36.81±0.05</td>
<td>36.81±0.06</td>
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</tr>
<tr>
<td>Day 22</td>
<td>36.67±0.04</td>
<td>36.70±0.05</td>
<td>36.74±0.05</td>
<td>36.72±0.05</td>
<td>36.74±0.05</td>
</tr>
<tr>
<td>$\Delta T_{es}$ sweat recruitment, °C</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Day 1</td>
<td>0.07±0.04</td>
<td>0.08±0.03</td>
<td>0.11±0.03</td>
<td>0.11±0.04</td>
<td>0.14±0.04</td>
</tr>
<tr>
<td>Day 8</td>
<td>0.05±0.04</td>
<td>0.10±0.05</td>
<td>0.10±0.04</td>
<td>0.11±0.05</td>
<td>0.10±0.05</td>
</tr>
<tr>
<td>Day 22</td>
<td>0.08±0.05</td>
<td>0.11±0.05</td>
<td>0.15±0.05</td>
<td>0.12±0.06</td>
<td>0.14±0.05</td>
</tr>
</tbody>
</table>

Values are means ± SE; $n=11$. $T_{sk}$, mean skin temperature; $T_{es}$, esophageal temperature; $\Delta$, change.
All local $\dot{m}_{sw}$, after 60 min of exercise, were elevated on both days 8 and 22 relative to day 1 ($P < 0.05$; Fig. 3A), but significant differences were only observed between days 22 and 8 for the forearm ($P < 0.05$; Fig. 3A). That is, for the remaining four skin sites, the heat adaptation-induced increments in local sweating were largely completed by day 8. While each local site displayed significant increments in steady-state sweating, between-site comparisons revealed that proportionately greater local $\dot{m}_{sw}$ increases were only evident on day 22 for the chest (relative to thigh) and the forearm (relative to thigh and forehead; $P < 0.05$; Fig. 3B). These observations imply that heat acclimation did indeed alter the distribution of sweating, but it did not elicit a generalized redistribution of steady-state sweating toward the limbs. Thus some regions experienced a greater relative increase in local steady-state sweating, but this acclimation-induced pattern did not reflect a systematic trunk-to-limb redistribution of sweat secretion.

**DISCUSSION**

The current experiment has provided a comprehensive evaluation of human sweating adaptations accompanying exercise in humid heat. We measured sweating responses during exercise-heat stress (cycling) from two trunk regions, both limbs and the head, without the confounding influence of pressure hidrosis, and we induced sweating before the commencement of exercise, negating nonthermal sudomotor influences. Furthermore, rigid and precise standardization of preexperimental diet, hydration status, baseline environmental conditions, posture, resting metabolic rate, and seasonal climatic influences were employed. Two novel findings have emerged. First, while interregional variations existed for the capacity to increase local, steady-state $\dot{m}_{sw}$, the previously reported, postacclimation trunk-to-limb sweat redistribution (15, 31, 37) was not supported by the current data. Second, while the $T_b$ thresholds for the initiation of sweating were reduced with heat acclimation, the absolute elevation in $T_b$, from its thermoneutral state to the initiation of sweating, remained unaltered. That is, the reduced $T_b$ sweat threshold precisely paralleled the decline in the thermoneutral resting $T_b$.

The primary focus of this project was to evaluate the possibility of a postacclimation redistribution of sweating. We observed a greater relative increase in forearm $\dot{m}_{sw}$ compared with that of the forehead. This pattern is consistent with the hypothesis of a trunk-to-limb sweat redistribution and data from the literature supporting the possibility (15, 31, 37). However, our current observation of a greater relative increase in chest $\dot{m}_{sw}$ compared with the thigh (Fig. 3B) is inconsistent with this pattern. Had a true peripheral displacement occurred, one would have expected to observe a greater relative elevation at both the forearm and thigh sweating than at each of the central sites (chest, scapula, forehead). This did not occur.

We have previously reported a greater acclimation-induced increase in $\dot{m}_{sw}$ at the forearm compared with the forehead (31), but sweating was not measured at any other skin region. Similarly, Shvartz et al. (37) measured $\dot{m}_{sw}$ at only three skin regions and did not statistically compare the relative increases among skin regions. Comparisons with the frequently cited acclimation data of Höfler (15) are also difficult and unconvincing because local $\dot{m}_{sw}$ were not reported, but pooled-mean $\dot{m}_{sw}$ for the trunk, head, arms, and legs were calculated. However, closer inspection of the original data revealed that only the contribution of the arms to total sweating was significantly elevated (17.4 $\pm$ 1.6 vs. 20.0 $\pm$ 1.3%; $P < 0.05$). The reported diminished contribution from the trunk was not significant (52.4 $\pm$ 2.6 vs. 48.8 $\pm$ 2.8%; $P > 0.05$), while the sweat responses of the head and legs were generally unaffected (8.7 $\pm$ 1.2 vs. 9.0 $\pm$ 1.6% and 21.5 $\pm$ 2.5 vs. 22.3 $\pm$ 1.6%, respectively; $P > 0.05$). The current investigation provides a more comprehensive assessment of local $\dot{m}_{sw}$ and demonstrated that not all limb skin regions will experience a greater relative elevation in $\dot{m}_{sw}$ relative to trunk regions after humid heat acclimation.

In the absence of neural evidence to support a differential sympathetic activation of recruited sweat glands among skin regions, which may lead to differences in gland training and hypertrophy, we suggest that the underlying mechanism for these local differences may be attributed to differences in sweat gland capacity. In the present investigation, sweat gland capacities (outputs) were not determined because maximal $\dot{m}_{sw}$ were not pharmacologically induced, and sweat gland densities were not assessed. However, if we accept the possibility that sweat glands can adapt, and that an adaptation spectrum exists, then we suggest that, before heat adaptation, local peak $\dot{m}_{sw}$ may fall at varying, but not identical, positions within this spectrum. Thus it is conceivable that glands falling toward the less adapted end of this spectrum will exhibit relatively greater flow increases after acclimation than will glands that fall within the more adapted region. Accordingly, during heat acclimation, all glands move, perhaps asymptotically, toward a position of superior adaptation, and the intersite variation in glandular capacities is reduced. We therefore hypothesize that skin regions farther away from their site-specific maximal glandular capacity (e.g., the forearm) will undergo the greatest increase in local sweat secretion. Such increases do not represent a systematic sweat redistribution, as previously implied, but are merely the consequence of all sites approaching their maximal capacities. Alternatively, it is possible that heat acclimation elicits a greater recruitment of sweat glands. However, the observations of Collins et al. (4) and Inoue et al. (16) are not consistent with this possibility, showing that heat acclimation does not facilitate recruitment of previously inactive glands, nor does it increase the density of active sweat gland.
not differ between HSTs, either at rest or at the sweat threshold, it is suggested that this offsetting of the sudomotor $T_b$ threshold primarily reflected a reduced resting deep body heat content, as reflected in the $T_c$. Because an equivalent $T_b$ displacement was required to initiate sweating across each HST, one may assume that the rate of heat gain at rest was probably similar before and after heat acclimation and was not associated with an earlier sweat onset. Thus, within the current experiment, heat acclimation did not modify heat storage before sweating, but it did affect the basal temperature from which that storage commenced.

It has previously been suggested that the sudomotor adaptations to heat acclimation take 10–14 days to become established (1). Indeed, Armstrong et al. (1) and Cotter et al. (6) failed to observe significant $m_{sh}$ elevations after short-duration heat acclimation, while others have reported a more rapid sudomotor adaptation (9, 14, 38). Our long-term, controlled-heat acclimation, while others have reported a more rapid attainment of some predetermined $T_b$.

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